The Founding Mitochondrial DNA Lineages of Tristan da Cunha Islanders

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ABSTRACT Genealogical histories show that the inhabitants of Tristan da Cunha are derived from a known number of founders. Using the transmission of mitochondrial DNA (mtDNA) from mother to offspring pairs, we traced the mtDNA types found in 161 extant individuals to five female founders. Although the historical data claimed that two pairs of sisters were among the founding females, mtDNA data showed support for only one pair of sisters. We also studied the fidelity of mtDNA transmission in conjunction with the genealogical data. We did not detect any mutations from 698 base pairs of sequence data from 75 individuals, which together accounted for 108 independent transmissions of mtDNA from mother to offspring. Based on this observation, we estimate that the mtDNA mutation rate is no more than one new mutation every 36 transmissions. These results indicate a high fidelity of maternal mtDNA transmission and support the utility of mtDNA in evolutionary and forensic studies. Am J Phys Anthropol 104:157–166, 1997.
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Tristan da Cunha is an island of volcanic origin, situated in the south Atlantic Ocean (Fig. 1). It spans an area of approximately 38 square miles, of which only some 3 square miles is suitable for settlement and agriculture. Tristan da Cunha was first discovered by the Portuguese in 1506; thereafter it was used by sailors as a stopover for replenishing supplies (Munch, 1945). In 1816 the British established a garrison on Tristan to prevent the French from rescuing Napoleon Bonaparte, who was in exile on St. Helena, some 1,350 miles to the north. In 1817 the garrison on Tristan was judged inconsequential to the security of Bonaparte on St. Helena and the British withdrew (Roberts, 1971).

On withdrawal of the garrison, Corporal William Glass, his wife (M.L.) and two children, plus two other men (John Nankivel and Samuel Burnell), were granted permission to remain on Tristan (Munch, 1945). This was the start of the first permanent settlement on the island. Only Glass and his wife contributed to the gene pool of the

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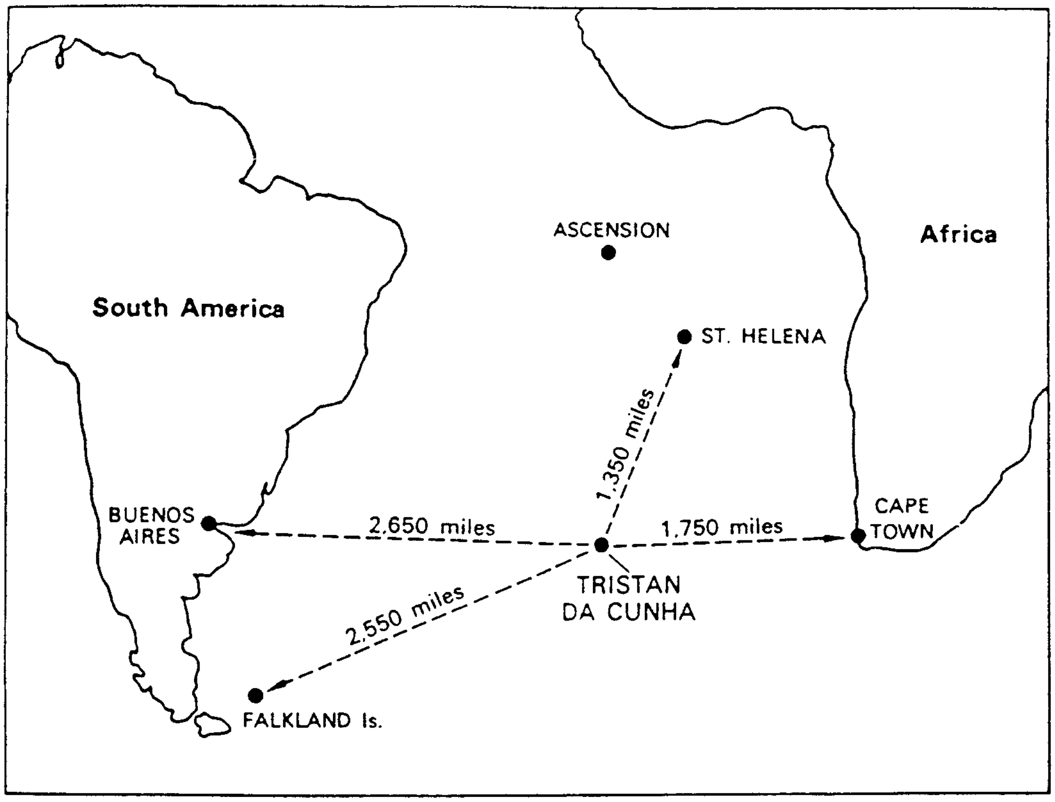


Fig. 1. Map of the South Atlantic showing the relationship of Tristan da Cunha to neighboring islands and land masses.

current population; the other two men left Tristan for Cape Town to sell the island's produce and did not return (Munch, 1945).

Subsequently, both male and female immigrants arrived on Tristan and the genealogy of the islanders has been reconstructed from family and church records (Munch, 1945; Roberts, 1968). Altogether, the current gene pool can be traced to 22 ancestors which includes seven females and 15 males. However, only seven surnames (Glass, Swain, Green, Rogers, Hagan, Repetto and Lavarello) remain in use among the 297 members of the population (Statistical Yearbook, 1994). The other eight men contributed to the gene pool but subsequently left the island, taking their surnames with them.

During the course of its history, 15 women brought genes to the population, but since some of these women and their descendants died or left the island, the nuclear gene pool of the present population is derived from only seven women (Roberts, 1968). According to the historical records, the first woman to arrive was M.L., who came from Cape Town in 1816. Subsequently, three women, among them two "sisters" (M.W. and S.W.) and one mother and daughter pair (M.W. and M.W.), arrived from St. Helena in 1827. S.P., also from St. Helena, arrived in 1863 and the second pair of sisters (E.S. and A.S.), of Anglo-Irish descent, arrived in 1908. Little is known about the women from St. Helena and Cape Town, but it has been suggested that they were of mixed ancestry (Munch, 1945).

Since mtDNA is maternally inherited and does not recombine (Giles et al., 1980; Zuckerman et al., 1984), it is possible to use the mtDNA types found in the present Tristan population to trace their ancestry to the founding female ancestors. The Tristan popu-

lation is also ideal to examine the fidelity of mtDNA transmission across several generations and to estimate the rate of human mtDNA mutation from family data in conjunction with the known genealogical and historical data.

In this study we used sequence-specific oligonucleotide (SSO) typing (Stoneking et al., 1991; Melton et al., 1995) to identify the mtDNA types found among 161 Tristan islanders sampled in 1982. Then, by judicious sampling of the Tristan genealogy, we estimated the mtDNA mutation rate from control region sequence data in 75 individuals who together accounted for 108 transmissions of mtDNA from mother to offspring pairs.

MATERIALS AND METHODS

Sample

We extracted DNA from the buffy coats of 161 individuals using the methods described by Sykes (1983) and the Isoquick DNA extraction procedure (Isoquick kit). We furnished Dr. Derek Roberts a list of the names and dates of birth of the subjects investigated in this study and he then provided us information depicting their maternal relationships from the genealogical data. Out of consideration for the living descendants of the founders, we have made reference to the founding females by using only their initials.

Methods

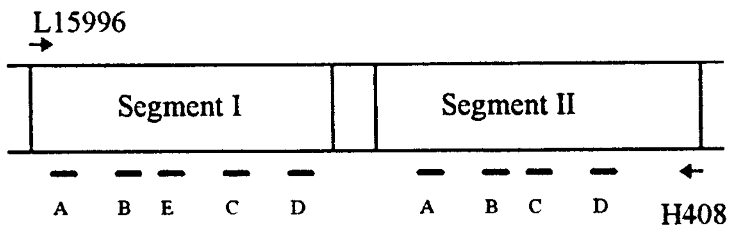
We amplified the 1.1 kilobase (kb) mtDNA control region using the polymerase chain reaction (PCR) procedure and primers L15996 and H408 (Fig. 2) as described by Redd et al. (1995). Approximately 5 µL of amplified products was fixed to nylon membranes (Biodyne) using the method described previously (Stoneking et al., 1991; Melton et al., 1995). MtDNA variation was surveyed within nine sequence regions by hybridization using 24 specific oligonucleotide (SSO) probes (Fig. 2) and detected using a non-radioactive chemiluminescent method (Melton et al., 1995). SSO-types were derived for each individual by combining the SSO variants obtained from regions IA, IB, IE, IC, ID, IIA, IIB, IIC and IID, in this order. Blanks were scored as zero (0) in the present nomenclature; this was obtained when none of the SSO probes designed for a particular region bound to the DNA, implying that one or more additional mutations were contained within this region.

We used the genealogical information to select the fewest number of individuals to maximize the number of maternal transmissions traced to each founding female. Altogether, we sequenced the two hypervariable segments of the control region from 75 of the 161 individuals examined in this study using the biotin/streptavidin method in conjunction with magnetic beads (Dynal), as described by Redd et al. (1995). DNA sequences were compared with the published reference sequence (Anderson et al., 1981) using GELIN (S. Sherry, the Pennsylvania State University). Pairwise comparisons of the sequences were made using the MEGA package (Kumar et al., 1993). The neighbor-joining (NJ) method of Saitou and Nei (1987) was used to study the phylogenetic relationship of the mtDNA lineages found on Tristan with those of Africans, Asians and Europeans (Vigilant et al., 1991; Soodyall, 1993). We used the bootstrap test (Felsenstein, 1975) with 1,000 replications to obtain statistical support for the tree.

RESULTS

MtDNA SSO-typing and control region sequence variation

We derived five unique mtDNA SSO-types from the sample of 161 Tristan islanders surveyed for mtDNA variation by the dot-blot method (Table 1). The Tristan population was polymorphic for eight of the nine regions, but was monomorphic for region IE (Table 1). Using the genealogical data we traced 46 individuals with mtDNA SSO-type 311112131 to S.W., 34 individuals with mtDNA SSO-type 121122231 to M.W., 25 individuals with mtDNA SSO-type 111111311 to the sisters E.S. and A.S., 11 individuals with mtDNA SSO-type 321312022 to M.L., and 45 individuals with mtDNA SSO-type 121312321 to S.P. Although S.W. and M.W. were described as "sisters" from the historical data, their descendants have distinctly different mtDNA SSO-types (Table 1). Their



SSO region	Nucleotide positions*	SSO variant	Mutation detected
IA	16118-16136	IA1	16126 T, 16129 G
		IA2	16126 C, 16129 G
		IA3	16126 T, 16129 A
IB	16211-16228	IB1	16217 T, 16223 C
		IB2	16217 T, 16223 T
		IB3	16217 C, 16223 C
IE	16244-16264	IE1	16247 A, 16261 C
		IE2	16247 A, 16261 T
		IE3	16247 G, 16261 C
IC	16300-16317	IC1	16304 T, 16311 T
		IC2	16304 C, 16311 T
		IC3	16304 T, 16311 C
ID	16357-16374	ID1	16362 T
		ID2	16362 C
IIA	68-83	IIA1	73 A
		IIA2	73 G
IIB	141-158	IIB1	146 T, 152 T
		IIB2	146 C, 152 T
		IIB3	146 T, 152 C
IIC	186-204	IIC1	195 T, 199 T
		IIC2	195 C, 199 T
		IIC3	195 T, 199 C
IID	240-258	IID1	247 G
		IID2	247 A

*Relative to the published reference sequence (Anderson et al. 1981)

Fig. 2. Schematic diagram of the mtDNA control region showing the approximate regions of binding of SSO probes within each hypervariable region, as well as a description of the mutations detected by each of the SSO probes used to detect specific mutations within the control region.

TABLE 1. Sequence specific oligonucleotide (SSO) type variation among Tristan da Cunha islanders showing the number of individuals with SSO-types traced to the founding females

Founding females	SSO variants detected within nine regions of the control region									Nos. with SSO-type
	IA	IB	IE	IC	ID	IIA	IIB	IIC	IID	
S.W.	3	1	1	1	1	2	1	3	1	46
M.W.	1	2	1	1	2	2	2	3	1	34
E.S. and A.S.	1	1	1	1	1	1	3	1	1	25
M.L.	3	2	1	3	1	2	0	2	2	11
S.P.	1	2	1	3	1	2	3	2	1	45
Total										161

TABLE 2. Sequence types found among Tristan da Cunha islanders traced to the founding females showing the variant positions and the mutations (boxed) detected by SSO-typing

			CR1										CR2								
				IA	IB					IC		ID	IIA	IIB	IIB	IIC	IIC		IID		
Founding female	Nos. with SSO-type	Nos. sequenced	16086	$\boxed{16129}$	$\boxed{16223}$	16230	16243	16263	16295	$\boxed{16311}$	16324	$\boxed{16362}$	$\boxed{73}$	$\boxed{146}$	$\boxed{152}$	$\boxed{195}$	$\boxed{199}$	204	$\boxed{247}$	263	294
Reference sequence[a]			T	G	C	A	T	T	C	T	T	T	A	T	T	T	T	T	G	A	T
S.W.	46	22	C	A	.	.	.	.	.	.	C	.	G	.	.	.	C	.	.	G	.
M.W.	34	16	.	.	T	.	.	.	T	.	.	C	G	C	.	.	C	.	.	G	.
E.S. and A.S.	25	14	.	.	.	.	.	.	.	.	.	.	.	.	C	.	.	.	.	G	.
M.L.	11	9	.	A	T	G	C	.	.	C	.	.	G	C	C	C	.	.	A	.	A
S.P.	45	14	.	.	T	.	.	C	.	C	.	.	G	.	C	C	.	C	.	G	.

[a]Anderson et al. (1981).

mtDNA types are too different to reflect mutation but instead suggest that they were not maternally related. The descendants of the other pair of sisters, E.S. and A.S., have identical mtDNA SSO-types (Table 1), confirming their sibling relationship from the historical data.

Sequence data confirmed the presence of variant sites detected by SSO-typing, but also revealed additional mutations at sites not screened for by the SSO-typing method (Table 2). In addition, we did not detect any variation among individuals from within any given lineage. To test the accuracy of SSO-typing, we inferred SSO-types from the sequence data; in every case the inferred SSO-types were identical to those actually observed.

Phylogenetic analysis

We used phylogenetic analysis to trace the geographic origins of the mtDNA types found in the Tristan population (Fig. 3). Although there is weak support for the phylogenetic relationship of mtDNA types in the NJ tree (Fig. 3), the sequence type traced to the sisters E.S. and A.S. (Table 2) cluster together with mtDNA types found in Europeans, a finding consistent with their presumed European ancestry. The sequence types of M.W. and S.W. (Table 2) cluster in branches of the NJ tree consisting of mtDNA types found in Chinese and European individuals (Fig. 3), though these branches are quite close to others containing African individuals. Also, the mtDNA sequence types found in descendants of S.P. cluster with sequences found in Europeans, while the mtDNA lineage traced to descendants of M.L. cluster in branches of the tree which are exclusively found in sub-Saharan Africans, particularly Khoisan peoples.

With the exception of the mtDNA type found in the sisters E.S. and A.S. of European origins, phylogenetic analysis does not unambiguously reveal the geographic origins of the other founding mtDNA types found in Tristan da Cuhna. However, a sample from St. Helena, and Cape Malay and Cape "Coloured" from South Africa would be ideal for these comparisons. Unfortunately, there are no data available from these populations at this time.

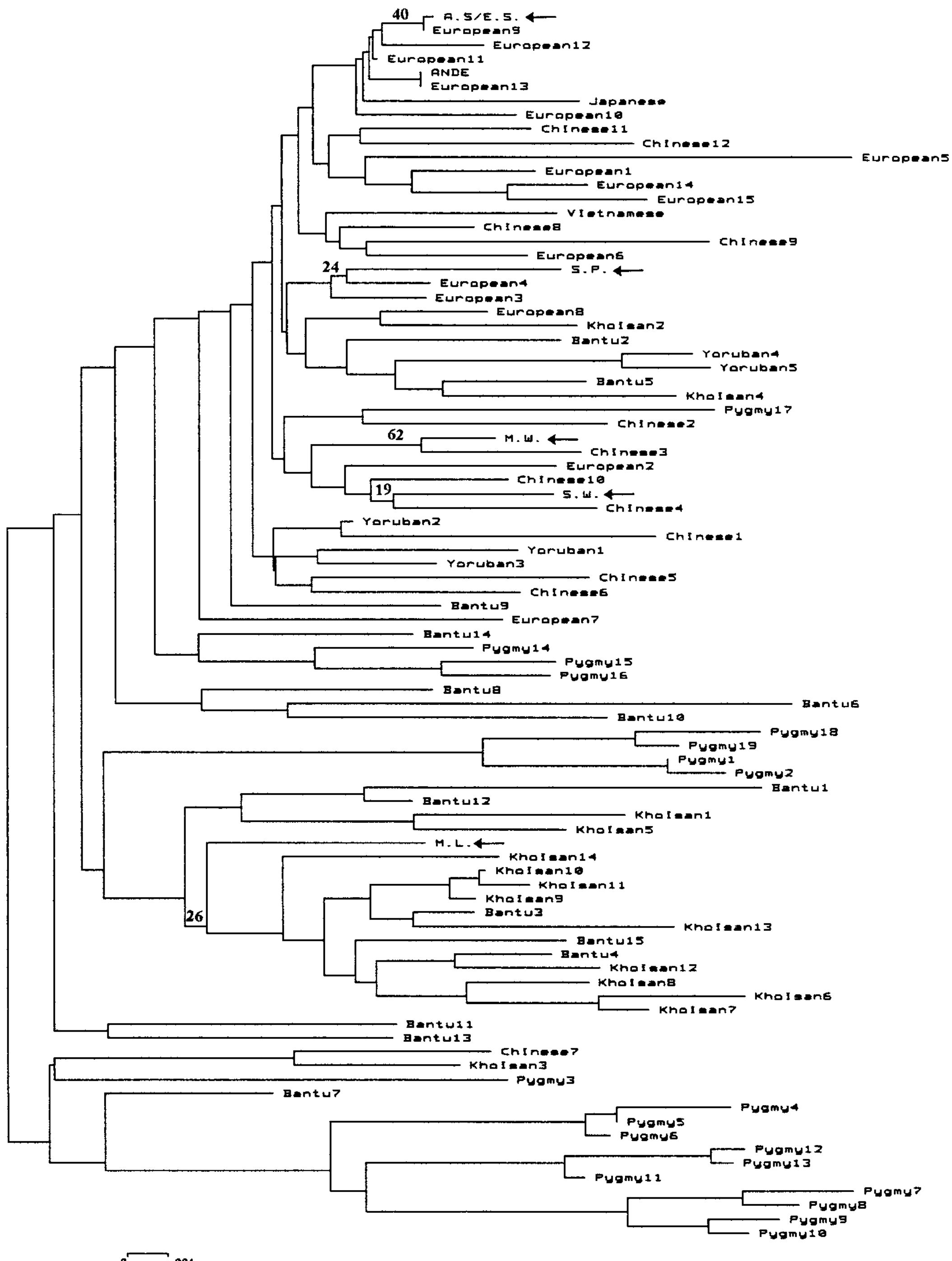


Fig. 3. Unrooted NJ tree showing the phylogenetic relationship of the mtDNA lineages found in Tristan da Cunha (denoted by arrows) to Africans, Asians and Europeans (Vigilant et al., 1991; Soodyall, 1993). The bootstrap support for the branches containing mtDNA sequences found among Tristan islanders are given as a percentage.

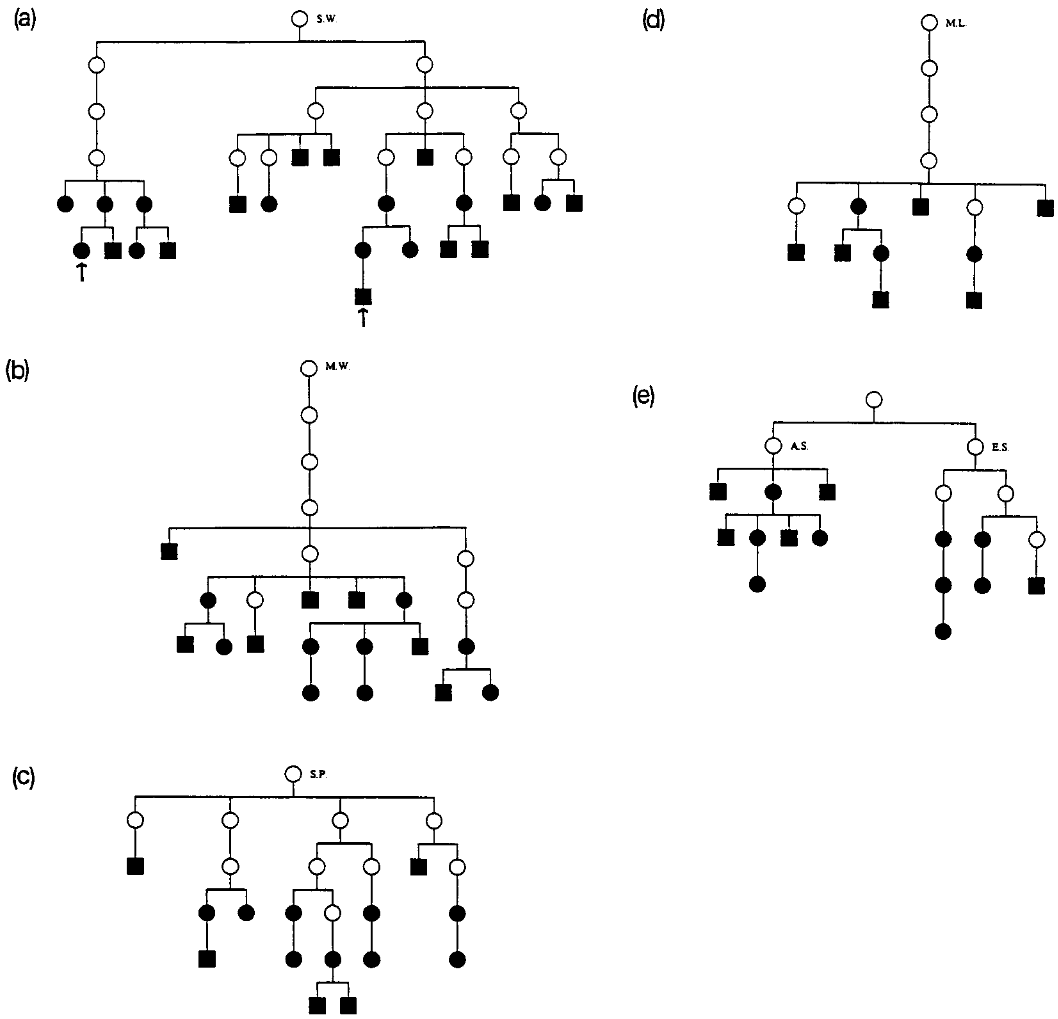


Fig. 4. Partial genealogies showing the transmission of mitochondrial DNA from extant individuals to the founding females of Tristan da Cunha: **(a)** S.W., **(b)** M.W., **(c)** S.P., **(d)** M.L. and **(e)** A.S. and E.S. Individuals from whom mtDNA sequence data was obtained are indicated by filled circles (females) and squares (males). Arrows indicate individuals separated by 11 transmissions of mtDNA.

Estimation of the mtDNA mutation rate

We used the genealogical data to estimate the number of mtDNA transmissions from each founding female to her descendants in our sample (Fig. 4). The greatest total number of mtDNA transmissions between any pair of individuals from whom sequence data were obtained was 11 and was found in the genealogy traced to S.W. (indicated by arrows in Fig. 4a). The number of transmissions and the number of mutations detected per mtDNA lineage are summarized in Table 3.

We did not, in fact, detect any mutation in any individual from a given lineage in a total of 108 transmissions. These data can be used to place an upper limit on the rate of mtDNA mutations. Assuming that mutations are independent events, the probability that no mutations would be observed in 108 transmissions is:

$$P = (1 - \mu)^{108}$$

where μ is the probability of a new mutation in a single transmission. Setting $P = 0.05$

TABLE 3. The number of mtDNA transmissions and the number of mutations detected along matrilineal lines derived from founding females of Tristan da Cunha

Founding females	Nos. of transmissions	Nos. of mutations
S.W.	35	0
M.W.	20	0
E.S. and A.S.	19	0
M.L.	11	0
S.P.	23	0
Total	108	0

and solving for μ gives an estimate for $\mu = 0.028$. This means that the mtDNA mutation rate is, with 95% confidence probability, at most one new mutation every 36 transmissions; in other words, if the mutation rate were higher than this, then the probability of observing at least one new mutation in 108 transmissions is greater than 95%.

DISCUSSION

MtDNA types in Tristan da Cuhna

Since the historical data show that the mitochondrial gene pool on Tristan da Cuhna is derived from seven founding females, including two pairs of sisters and one mother-daughter pair, we would expect to find a maximum of four mtDNA types in the Tristan population. Instead, we have identified five different mtDNA lineages by SSO-typing (Table 1) and DNA sequencing (Table 2). The reason for the discrepancy is that the descendants of one pair of "sisters," S.W. and M.W., have different mtDNA types.

It is unlikely that the seven nucleotide differences found in descendants of S.W. and M.W. (Table 2) are the result of new mutations which occurred on Tristan following the arrival of the "sisters" in 1826. One possibility is that one of the "sisters" could have inherited her father's mtDNA. Since genetic material is not available from the latter, we cannot verify whether paternal inheritance has contributed to this discrepancy, though this seems unlikely for reasons discussed later. Another explanation for the discrepancy in the mtDNA data found in descendants of S.W. and M.W. is that they had the same father but different mothers. Since we do not have DNA from S.W. and M.W. we cannot verify their genetic relationship using autosomal markers. It is also possible that "sisters" in this instance is a kinship term with non-western meaning, and may have no biological meaning.

Both S.W. and M.W. were described as being of "mixed" ancestry (Munch, 1945), but the origin of their mtDNA types cannot be confirmed from these data. There is, however, some suggestion based on the pattern of clustering of mtDNA sequence types (Fig. 3) that S.W. and M.W. may have inherited their mtDNAs from Asian ancestors. This analysis does not rule out the possibility of European or African ancestry, however.

M.L. married William Glass in Cape Town before moving to Tristan in 1816. According to some accounts, M.L., because of her physical appearance, was described as a Cape Creole (Munch, 1945). The mtDNA SSO-type 321312022 traced to M.L. has been found in 24 Africans (Soodyall, unpublished). In fact, the mtDNA sequence type traced to M.L. clusters with mtDNA types found in Khoisan and Bantu-speaking individuals (Fig. 3). It seems likely, therefore, that M.L. was also of mixed ancestry and there is a strong suggestion that her maternal ancestor was of African origin.

The mtDNA SSO-type 121312321 was found among descendants of S.P., who arrived in Tristan from St. Helena in 1863. This SSO-type has been found in two Negroid individuals from Africa and 13 Malagasy (Soodyall, unpublished). The clustering of this mtDNA in the NJ tree (Fig. 3), however, suggests a close relationship with Europeans. Closer examination of the sequence data reveals that descendants of S.P. and the two Europeans differ at several sites, but also have some common mutations which may contribute to the clustering pattern in the NJ tree (Fig. 3). Therefore, mtDNA data could not unambiguously resolve the geographic origin of the mtDNA type contributed by S.P.

The descendants of E.S. and A.S. all have the same mtDNA SSO-type (111111311), confirming their sibling relationship from the historical data. In addition, the European ancestry of these two sisters is also supported by the clustering of their mtDNA sequence type (Fig. 3).

Fidelity of mtDNA transmission

The maternal (uniparental) mode of transmission of mtDNA has been challenged by recent findings of biparental transmission of mtDNA in several animal species (Kondo et al., 1990; Hoeh et al., 1991; Gyllensten et al., 1991; Zouros et al., 1992, 1994). We did not detect any mutations in any lineage derived from a founding female for a number of mother to offspring transmissions (Table 3), even in a lineage separated by 11 generations (Fig. 4a). There was also no evidence of paternal leakage in families where the father had a different mtDNA type from the mother. Our findings demonstrate a high fidelity of mtDNA transmission from mother to offspring over successive generations, confirming that the inheritance of human mtDNA is strictly maternal.

MtDNA mutation rate

Current estimates of the mtDNA mutation rate are based on indirect methods from evolutionary studies (Vigilant et al., 1991; Stoneking et al., 1992; Tamura and Nei, 1993; Horai et al., 1995). For example, using the rate of mutation of 11.81% per million years (Stoneking et al., 1992), we estimated that one mutation occurs every 600 transmissions (assuming a generation time of 20 years). This is an average rate over the two hypervariable regions of the control region, has a large standard error and moreover is probably an underestimate for some nucleotide positions that are suspected to evolve more rapidly than the average rate (Tamura and Nei, 1993; Wakely, 1993).

In this study we estimated the mtDNA mutation rate directly from family data in the Tristan population. Since we did not detect any mutations in 108 transmissions (Table 3), we estimate, with 95% confidence probability, that the mutation rate is not greater than one in every 36 transmissions, which is equivalent to one in every 720 years (assuming a generation time of 20 years). However, Howell et al. (1996) have recently estimated a slightly higher mutation rate, one every 25 generations, using control region sequence data from a family with Leber hereditary optic neuropathy (LHON). These authors suggest that 1) abnormal mitochondrial metabolism may accelerate the rate of mutation in families with mitochondrial disorders, and 2) the rate of evolution may be higher in some mtDNA lineages than others.

In contrast to the study by Howell et al. (1996), we have examined a large number of transmissions (the largest of any study to date) in a population that does not have any mtDNA disorders (so far as we know from the historical records) and found no new mutations. Therefore, additional family studies are needed to estimate more accurately the rate of human mtDNA mutation and to determine if there is variation in this rate among families. Overall, our results indicate a high fidelity of maternal mtDNA transmission, confirming the utility of mtDNA for evolutionary studies (Stoneking, 1993) and in forensic applications involving individual identification (Wilson et al., 1993).

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